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v.2.0

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To: Sundhedsministeriet, Sundhedsstyrelsen and Statens Serum Institut
cc: Working Group for Tg (v/FVST)
cc: **Whom it may concern.**

**Covid-19 cytokine findings match those of toxoplasmosis. Action must be taken. Authorities can not keep ignoring that Covid-19 and toxoplasmosis has a huge overlap in virtually ALL aspects. Ignoring this could be fatal if the data points are correct.**

This document is a supplement to our previous documents on "Covid/Toxoplasma correlations" of March April, May and June. All documents can be found on the homepage of the Parliament of Denmark. **Please refer to these for a comprehensive list of overlaps between Toxoplasma and Covid-19.**

For further information on Toxoplasma, please see this overview article.

Covid-19 keeps surprising as the Coronavirus family usually is related to "common cold". However Covid-19 displays a set of completely new symptoms for this type of virus and its general pathology;

> "..a clear picture is elusive, as the **virus acts like no pathogen humanity has ever seen**" (Science, April 2020)

***There is an almost perfect overlap between the symptoms presented in Covid and in acute toxoplasmosis. This overlap extends to outcomes, suggested treatments, and pathways for activation of acute toxoplasmosis.***

Toxoplasma is – by far – the most prevalent infection among humans, and its distribution and proliferation **are clearly mirrored in Covid-19 cases, pathology and fatalities**.

In this research note we elucidate the exact overlap in the cytokines involved in Covid-19 and those involved in the pathology of toxoplasmosis:

**COVID**:

"Thirty-eight out of the 48 measured cytokines in the plasma of 2019-nCoV-infected patients were significantly elevated compared to healthy individuals. **Seventeen cytokines were linked to 2019-nCoV loads**. Fifteen cytokines, namely M-CSF, IL-10, IFN-$\alpha$2, IL-17, IL-4, IP-10, IL-7, IL-1ra, G-CSF, IL-12, IFN-$\gamma$, IL-1$\alpha$, IL-2, HGF and PDGF-BB, were strongly associated with the lung-injury"

https://academic.oup.com/nsr/article/7/6/1003/5800998

**Toxoplasma**:
As shown here, there is a perfect match:

**"There is strong experimental evidence that the cytokines play a major role in the pathogenesis of Toxoplasma gondii** and manipulation of these cytokines can put forth a beneficial or damaging effect on the host and thus modulate the disease pathology.

The importance of interferon gamma (IFN-$\gamma$) was clearly demonstrated when the neutralization of interleukin (IL) IL-12 and of IFN-$\gamma$ using anti-IFN-$\gamma$ & anti-IL-12 monoclonal antibodies (MAb) resulted in reactivation and loss of control on the parasite's multiplication leading to disease. Cytokine IL-27 was deemed as an endogenous suppresser on IL-17, the inflammatory cytokine that is capable of enhancing the inflammatory response in the brain. Although Toxoplasma is an intracellular pathogen thereby requiring a cell mediated immune response from the host, the cytokine IL-10 protects the infected mice from an exaggerated cellular immune response by inhibiting the production of pro inflammatory cytokines: IL12, IFN-$\gamma$, and TNF-$\beta$. Following Toxoplasma infection the host triggers a sequential balanced cytokine response to limit the infection and the disease pathology. However, cytokines can sometimes exert a negative effect on the host and augment the disease leading to severe irreversible tissue damage."

https://www.semanticscholar.org/paper/Toxoplasmosis-%3A-Role-of-Cytokines-in-Disease-%26-Iqbal-Al-Awadhi/217d5b243d27c4f34c3c591272cf2708d2173acc

"The present study shows both a human platelet activation by free tachyzoites of T. gondii and human platelet-mediated cytoinhibition of T. gondii intracellular growth invitro, in the absence of antibodies. The results suggest a prominent role of platelet-derived growth factor (PDGF) in this phenomenon."

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1904904/

"All 4 canonical pathways altered by type II strain infection involved signaling (see Data Set S6), and genes within these pathways were generally upregulated. Among them, the prolactin and hepatocyte growth factor (HGF) signaling pathways were the most enriched."

https://iai.asm.org/content/79/3/1363

"The findings substantiate a separate, T. gondii-induced pathway of astroglia activation characterized by the release of IL-1 which **may drive local inflammatory reaction both at initial infection of the brain and during reactivating toxoplasmosis.**"

"Of these cytokines, M-CSF and GM-CSF proved to regulate growth and differentiation of microglia; IL-1, IL-6, and TNF-a those of astrocytes."

https://onlinelibrary.wiley.com/doi/abs/10.1002/eji.1830270633

"Culture supernatants from spleen cells derived from IL-4-deficient mice contained significantly more gamma interferon than those derived from IL-4+/+ mice at day 7 postinfection. Conversely, IL-10 production was significantly greater from the spleen cells derived from wild-type mice at day 28 postinfection."

https://iai.asm.org/content/64/3/897.short

"IP-10 Is Critical for Effector T Cell Trafficking and Host Survival in Toxoplasma gondii Infection."

https://www.sciencedirect.com/science/article/pii/S1074761300802009

"IL-7 stimulates protective immunity in mice against the intracellular pathogen, Toxoplasma gondii."

https://www.jimmunol.org/content/157/5/2103.short

"Toxoplasma gondii Induces Granulocyte Colony-Stimulating Factor (G-CSF) and Granulocyte-Macrophage Colony-Stimulating Factor Secretion by Human Fibroblasts: Implications for Neutrophil Apoptosis."

https://iai.asm.org/content/70/11/6048.short

"Pro-inflammatory cytokines, such as IL-1 $\alpha$, IL-1 $\beta$, IL-6, and IL-10 **increased rapidly at week 1** post-infection (PI) and **peaked at week 3."**

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3121066/

"A study was undertaken to determine the kinetics and mechanism of the observed downregulation

of interleukin 2 (IL-2) production during experimental murine toxoplasmosis"

https://iai.asm.org/content/62/7/2908.short

**There is no protocol in place that tests patients and/or deceased for the presence of Toxoplasma tachyzoites/acute toxoplasmosis. Why is that?**

All quotes above are from peer-reviewed material. We will be happy to share this with anyone willing to assist in the elucidation of the above correlations.